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Clinical letter

Seizure-Induced Myocardial Stunning: A Possible Cardiac Link to Sudden Unexpected Death In Epilepsy (SUDEP)

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1. Introduction

Transient left ventricular dysfunction complicating neurological conditions has been well described. Although most commonly linked to subarachnoid hemorrhage, "neurogenic stunned myocardium" has also been reported to complicate metastatic brain tumors, subdural hematoma, hydrocephalus and ischemic strokes.[3]

We report a case of transient cardiomyopathy complicating generalized tonic-clonic seizures.

2. Case presentation

A 75-year-old female presented to the hospital after a witnessed seizure. She had two generalized tonic-clonic seizures one year prior to this event, which were attributed to cerebrovascular disease. The seizures were controlled with levetiracetam. Her medical history also included two strokes, hypertension and coronary artery disease with prior stent placement to the right coronary artery and normal left ventricular ejection fraction of 55%.

On the day of admission, while having breakfast, she experienced a generalized tonic-clonic seizure associated with urinary incontinence. Nothing in the history suggested a focal origin of the seizure. The patient was brought to the hospital when the seizure was followed by mild confusion. The patient's daughter reported that the patient missed the morning dose of levetiracetam. The family did not recall the patient complaining of chest pain, shortness of breath, palpitations or any cardiovascular symptoms, before or after the seizure.

In the emergency department, the patient had no complaints. Physical examination, other than confirming mild confusion, was normal. The blood pressure was 122/70 mmHg and the heart rate was 79 BPM. At the hospital, the patient had another generalized seizure that was not preceded or followed by focal neurological deficits. During the postictal period, an EKG was done showing ventricular tachycardia (Fig. 1) that terminated spontaneously; other than this episode, the telemetry showed no arrhythmias prior to, during or after the seizure.

Initial laboratory studies of electrolytes, complete blood count and cardiac enzymes were within normal limits. CT of the head showed an old left frontal infarction with no acute changes. Limited echocardiogram was performed to evaluate the ventricular tachycardia. It revealed an ejection fraction of 30% with multiple wall motion abnormalities that did not follow any coronary artery distribution (Fig. 2).

In view of systolic dysfunction, wall motion abnormalities, and the episode of ventricular tachycardia, urgent cardiac catheterization was performed. The stent in the right coronary artery was patent and the left anterior descending artery had 50% stenosis with normal fractional flow reserve which confirmed that the lesion was not hemodynamically significant. No intervention was performed as these findings did not explain the drop in ejection fraction.

Follow-up echo 4 days later, showed normal ejection fraction of 55% with resolution of the wall motion abnormalities.

3. Discussion

Patients with epilepsy have higher mortality rates and 24-fold higher risk of sudden death compared with the general population.[1] Up to 18% of all deaths in patients with epilepsy have been attributed to the Sudden Unexpected Death in Epilepsy (SUDEP) syndrome.[2] Epilepsy affects over 50 million people worldwide, and a better understanding of the neurological disorder and the mechanism of sudden death in these patients is essential.

Cardiac mechanisms causing SUDEP have been postulated as being related to bradycardic or tachycardic rhythms and/or acute

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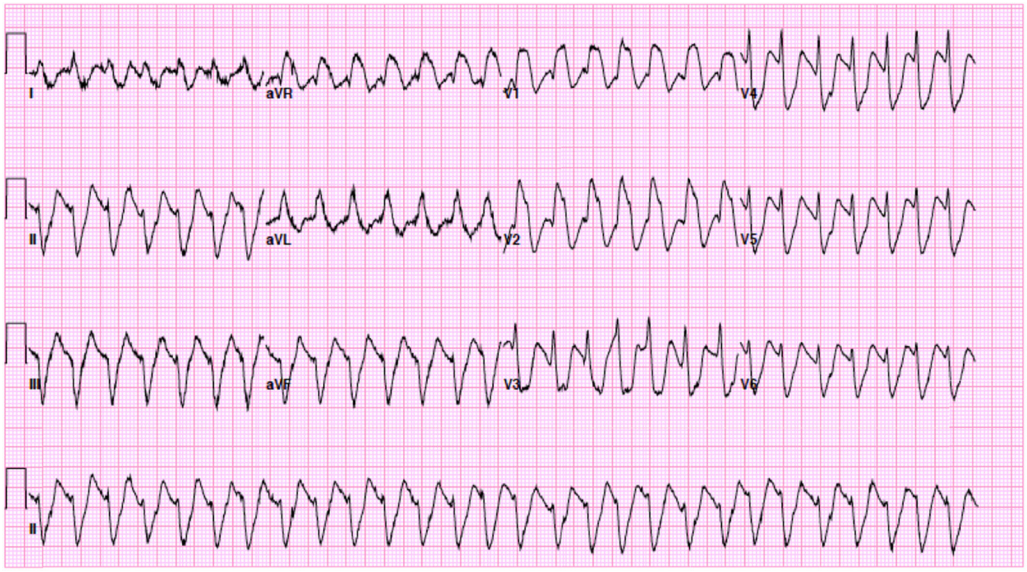

Fig. 1. An EKG during the immediate post-ictal period showing ventricular tachycardia.

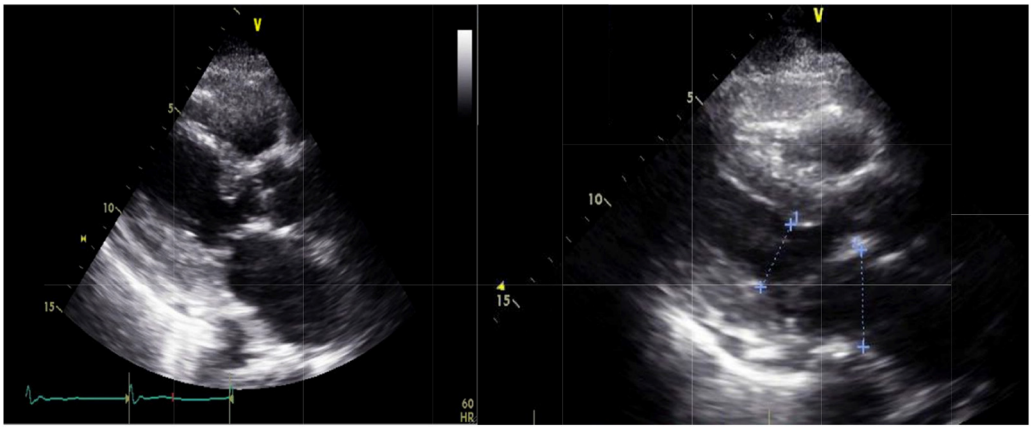

Fig. 2. On the left: an end-systolic frame when the ejection fraction was 30%. On the right an end-systolic frame on day 4 of hospitalization when the ejection fraction improved to 65%. Note the dramatic improvement in the inward wall motion during systole.

coronary ischemic events. The higher risk of arrhythmias[4] has been variably attributed to genetic predisposition in epileptic patients, pro-arrhythmic effects of anti-seizure medications, and to high sympathetic tone associated with seizures.[1]

Our patient presents the possibility that transient left ventricular (LV) systolic dysfunction, with or without malignant arrhythmias, may contribute to the increased morbidity and mortality. This syndrome may be akin to the transient neurogenic stunned myocardium (NSM) seen with subarachnoid hemorrhage (SAH). It is believed that extreme sympathetic discharge following subarachnoid hemorrhage contributes to transient LV systolic dysfunction. A similar sympathetic storm has been reported to occur with seizures likely explaining the transient LV systolic dysfunction seen in our patient. This possibility is supported by the fact that myocardial function normalized 4 days after the seizures were controlled.

NSM has many similarities to Takotsubo cardiomyopathy. They probably represent two different presentations of transient cardiomyopathy triggered by excessive sympathetic discharge. Our patient did not demonstrate classic symptoms, EKG or echocardiographic findings associated with Takotsubo syndrome.

There have been a few case reports of seizure-induced cardiac stunning but those reports did not rule out cardiac ischemia with coronary angiography. Our patient underwent coronary angiography which, in combination with the fractional flow reserve, excluded an ischemic etiology. Moreover, the non-coronary distribution of the wall motion abnormalities renders unlikely the hypothesis of ischemia secondary to coronary spasm.

Our case illustrates that seizures can cause transient LV systolic dysfunction. Most cases of SUDEP occur in the setting of seizure activity. Therefore, unrecognized myocardial stunning with or without malignant arrhythmias could be a factor in the higher rates of mortality observed in patients with epilepsy.[2]

The case we present has implications for possible prevention of sudden death in patients with seizures. Beta-blocker therapy in patients with subarachnoid hemorrhage has been associated with reduced incidence of cardiomyopathy and improved survival. It is possible that the use of beta-blockers may reduce the deleterious effect of the sympathetic storm during seizures thereby decreasing the risk of SUDEP. Future studies are needed to test this hypothesis.

Conflicts of interest

None declared.

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